Kindly replace the paragraph beginning at page 3, lines 8-19 with the following new paragraph:

Another mechanism that may contribute to the inefficient induction of tumor-reactive T cells is suggested by the "two-signal" model for lymphocyte activation. This model was originally proposed for B lymphocytes (Bretscher and Cohn, Science 169:1042-1049, 1970, incorporated herein by reference) and later as an explanation for why antigens expressed on cells of nonhematopoietic origin are ineffective at inducing transplant rejection (Lafferty et al., Ann. Rev. Immunol. 1:143-173, 1983, incorporated herein by reference). A two-signal model has now been proposed for all lymphocytes (Janeway, C.A., Jr., Cold Spring Harbor Quant. Biol. 54:1-13, 1989; Nossal, G. J. V., Science 245:147-153, 1989; and Schwartz, Cell 57:1073-1081, 1989, each incorporated herein by reference). According to this model, optimal stimulation and effective antigen-specific clonal expansion of lymphocytes require both a primary, antigen-specific signal, and a secondary, "co-stimulation" signal.

Kindly replace the paragraph at page 7, lines 3-11 with the following new paragraph:

In alternative aspects of the invention, the peptide or protein antigen may incorporate a T cell epitope of a tumor antigen or antigen of a viral or non-viral pathogen. In more detailed aspects, the peptide or protein antigen incorporates an epitope from a tumor antigen, for example the proteins (or products encoded by) p53, *ras*, *rb*, *mcc*, *apc*, *dcc, nfl,* VHL, MEN1, MEN2, MLM, Her-2neu, CEA, PSA, Muc1, Gp100, tyrosinase, and MART1. Alternatively, the peptide or protein antigen may comprise an epitope of a viral antigen, for example an antigen of human immunodeficiency virus (HIV), hepatitis B virus (HBV), hepatitis C virus (HCV), herpes simplex virus (HSV) or human papilloma virus (HPV).

Kindly replace the paragraph at page 13, lines 13-26 with the following new paragraph:

Therefore, mutant *ras* peptides serve as particularly useful vaccine agents to elicit anti-cancer immune responses according to the methods of the invention. In this context, *Ras* p21 is an intracellular protein subject to antigen processing and presentation by MHC molecules. Specific CD4+ and CD8+ T lymphocytes that can recognize a single *ras* mutation have been described. Murine experiments have shown that T lymphocytes specifically immunoreactive against mutated *ras* peptides have the ability to lyse target cells that endogenously express the same point mutated *ras* gene. These lytic T cells display cytotoxic activity of both CD4+ (Th1 subtype) and CD8+ subsets (Abrams et al., Eur. J. Immunol. 25:2588-2597, 1995; Peace et al., J. Immunother. 14:110-114, 1993; Peace et al., J. Exp. Med. 179:473-479, 1994; and Skipper et al., J. Exp. Med. 177:1493-1498, 1993, each incorporated herein by reference). Furthermore, induction of anti-*Ras* CTLs by vaccinating mice with recombinant mutant *ras* proteins has led to the rejection of syngeneic tumor cells bearing the corresponding mutation (Fenton et al., J. Natl. Cancer Inst. 85:1294-1302, 1993, incorporated herein by reference).

Kindly replace the paragraph beginning at page 19, line 8 with the following new paragraph:

Additional HIV peptide antigens (designated by source protein/amino acid sequence/and position) for use within the invention include P21 (gp120/QIDSKLREQFGNNK/410-429) (SEQ ID NO. 38); (gp120/GSDTITLPCRIKQFINMWQE/644-658) (SEQ ID NO. 39); P41 (gp41/NYTSLIHSLIEESQN/664-678) (SEQ ID NO. 40); P42 (gp41/EQELLELDKWASLWN/787-801) (SEQ ID NO. 41); P47 (gp41/RIVELLGRRGWEALK/172-196) (SEQ ID NO. 42); (pol(rt)/IETVPVKLKPGMDGPKVKQWPLTEE/325-349) (SEQ ID NO. 43); (pol(rt)/AIFQSSMTKILEPFRKQNPDIVIYQ/342-366) (SEQ ID NO. 44);

(pol(rt)/NPDIVIYQYMDDLYVGSDLEIGQHR/359-383) (SEQ ID NO. 45); (pol(rt)/DLEIGQHRTKIEELRQHLLRWGLTT/461-485) (SEQ ID NO.46); (pol(rt)/PLTEEAELELAENREILKEPVHGVY/495-519) (SEQ ID NO. 47); and (pol(rt)/EIQKQGQGQWTYQIYQEPFKNLKTG/265-279) (SEQ ID NO. 48) (Sequence numbers for gp120 and gp41 are from Ratner et al., Nature 313:277-284, 1985, and sequence numbers for pol and gag proteins from Sciliciano et al., Cell 54:561, 1988, and Walker et al., Proc. Natl. Acad. Sci. USA 86:9514, each incorporated herein by reference).

Kindly replace the paragraph beginning at page 30, line 16 with the following new paragraph:

Analogs of peptide or protein antigens and co-stimulatory proteins may be readily constructed, e.g., using peptide synthetic techniques well known in the art such as solid phase peptide synthesis (Merrifield synthesis) and the like, or by recombinant DNA techniques well known in the art. Techniques for making substitution mutations at predetermined sites in DNA include for example M13 mutagenesis. Manipulation of DNA sequences to produce substitutional, insertional, or deletional variants are conveniently described elsewhere such as Sambrook et al., 1989, *supra*. In accordance with these and related teachings, defined mutations can be introduced into a native peptide or protein antigen or co-stimulatory protein to generate analogs of interest by a variety of conventional techniques, e.g., site-directed mutagenesis of a cDNA copy of the peptide or protein antigen or co-stimulatory protein. This can be achieved through and intermediate of single-stranded form, such as using the MUTA-gen® kit of Bio-Rad Laboratories (Richmond, CA), or a method using the double-stranded plasmid directly as a template such as the Chameleon® mutagenesis kit of Strategene (La Jolla, CA), or by the polymerase chain reaction employing either an oligonucleotide primer or a template which contains the mutation(s) of interest. A mutated subfragment can then be assembled into a complete analog-encoding cDNA. A variety of other mutagenesis techniques are known and can be routinely adapted for use in producing the mutations of